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CASES
OF
PARAPLEGIA.

BY
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C A S E S
OF
P A R A P L E G I A .
SECOND SERIES.

WILLIAM GULL, M.D.

THE following cases of paraplegia, with those in the 'Reports' for 1856, though a miscellaneous contribution, may perhaps serve for reference in the absence of a more systematic treatise on the subject.

The labours of Lockhart Clarke, and Lenhossék on the minute structure of the nervous centres in health, cannot fail to give a new impulse to a more exact knowledge of the pathological changes to which they are subject. Something in this direction has been attempted. Those who are acquainted with the results of minute anatomy, as applied to the cord, will admit that we may now hope for an exhaustive morbid anatomy of it, exhaustive, at least, so far as to enable us to determine the state of the ultimate tissue.

Case xvii goes far to establish an important point in the pathology of paraplegia, namely, that the spinal centres may be paralysed without anatomical change of their structure. If this were certain it could not fail to give a new direction to our inquiries, and lead us with more earnestness to investigate the nervous substance by other means than the microscope. Dr. Sankey's observation on the variable specific gravity of the brain, lets in some light in this direction. It is from an increased knowledge of 'atomical,' as distinguished

from 'anatomical' conditions, that we may hope for future advances in nervous pathology.

CASE XVIII presents a not uncommon history of chronic inflammatory degeneration of the columns of the cord almost latent up to a certain point, and then accompanied by a sudden aggravation of the symptoms. It may offer an occasion to remark that in diseases of the nerve-substance, acuteness of effects is no evidence of acuteness of the lesion producing them. In the brain this is notoriously true, for every one knows that a sudden hemiplegia may result from local changes of the slowest and most passive kind. The same occasionally occurs in the cord. The bearing of this on diagnosis and treatment is obvious.

CASE XIX is a remarkable instance of the limitation of disease to the posterior columns. The lesion was of the same character as in the preceding case. The symptoms confirm the theory of Dr. Todd that the posterior columns are the channels through which the voluntary movements are co-ordinated. In this case there was not paralysis, but a want of controlling power. There was only a slight affection of sensation, proving also that the posterior columns are not mainly subservient to the sensory function.

CASE XX presented at the bed-side a rare symptom in paraplegia, namely, paralysis of both seventh nerves. This prevented the pronunciation of the labial parts of speech, and led to a suspicion of brain-disease until the kind of defect was pointed out. This accident was explained by the condition of the medulla oblongata.

As it has been just remarked, on the one hand, that the character of a lesion of the nervous substance is not to be inferred from the acuteness of the symptoms in respect to their development in time, so it may be added, on the other, that the amount of the lesion is not necessarily in proportion of the gravity of the symptoms. A very small amount of anatomical disease, or, as we seem to have proved in Case XVII, not enough to be recognised, may produce fatal effects. The degree of positive lesion was appreciable in this case, but it was in amount trifling. It was its seat which gave it its importance. It is in the treatment that we need to bear these truths in mind, as no doubt there is a proneness in the mind, as before said, to

estimate the activity and violence of a disease by the rapid development and danger of the symptoms, and, consequently, to aggravate it by too heroic interference.

In Case *xxi* the limitation of the disease at its onset to the right side of the cord, and the suddenness of the early symptoms are the chief points of pathological interest.

The occurrence of erysipelatous inflammation from the incautious application of heat, is an accident to which paraplegic patients are notoriously exposed.

It does not seem unimportant to draw attention to the difference between capillaries mechanically incrustated with fat-globules as the result of disease of the tissue in which they lie, and that form of fatty degeneration which is precursory of atrophy.

Case *xxii* shows that the substance of the cord may be damaged by a violent exertion, without any affection of the bones, ligaments, or membranes of the spine. Whilst such injuries have an immediate interest to the surgeon, they have not less a deferred interest to the physician, who is often called upon to treat the subsequent effects. It is on this account that I have recorded this case and Cases *xxiii* and *xxiv*. In Case *xxiii* there was bruising and ecchymosis of the posterior columns and of the grey matter, followed by hyperæsthesia of the parts below. In Case *xxiv* there was, first paraplegia from concussion of the cord, recovery after a few hours; and subsequently, fatal paraplegia from extravasation of blood outside the theca-vertebralis along the spinal canal.

Cases *xxv* and *xxvi* are instances of progressive muscular atrophy from chronic disease of the cord. The early symptoms of such cases are like those which come on in lead-poisoning. The wrists drop and the hands become weak. It seems hardly necessary to assert that such symptoms are not pathognomonic of the presence of lead, as some have stated, for it must be obvious to any one who will consider the matter that in chronic affections of the cord in the cervical region the disease is not always so uniform in its seat and extension as to affect the muscles in the same order. In one case the arms may waste generally and equally throughout; in another, the scapular muscles and those of the shoulder may be first affected; and in a third, the interossei of the fingers,

the short muscles of the thumb, and the extensors of the wrists may first fail. The relation of the nerves of the brachial plexus to the cervical enlargement of the cord partly explains these differences, and what remains obscure seems to require for its elucidation only a more accurate investigation of the distribution of the lesion through the cord in particular instances.

Case xxvii and xxviii are recorded for the purpose of pointing out the occurrence of acute rheumatic symptoms after spinal lesions. Mere pain in the joints and limbs, generally, is not what is here meant. It is too common an error to account for obscure pains by calling them 'rheumatic,' to need any remark. The symptoms referred to are commonly regarded as pathognomonic of a rheumatic state, namely, swelling and redness of the joints; profuse acid sweats; high coloured, scanty urine, depositing urates; &c. Our ignorance of the essential nature of acute rheumatism, prevents our asserting or denying that it may have its origin in a disturbance of the nervous force, but certainly a condition apt to be confounded with it does so arise. In practice it cannot be an indifferent matter, whether according to popular pathology we set before us, as the object of our treatment, the elimination of a materies morbi, or a lesion of the nervous centres.

Case xxix is remarkable for its clinical history, and the apparent contradictions which misled the diagnosis for the first two or three years. It was a case of chronic thickening of the spinal membranes implicating and destroying the posterior roots of the nerves of the brachial plexus. The disease was for some time so limited as to produce no other symptom than numbness of the left arm.

The test by galvanism, proposed by Duchenne, was entirely fallacious. This excellent author, in a *resumé* of his deductions on what he terms "faradisation," (electro-magnetism,) applied to pathology, gives the following conclusions in respect to hysteric paralysis:¹ 1. Electro-muscular-contractility is normal in hysterical paralysis. 2. Electro-muscular-sensibility is on the contrary generally diminished, or altogether absent.

¹ 'De l'Electrisation localisée,' p. 530.

3. Lastly, voluntary movements may be intact, notwithstanding the diminution or loss of the electro-muscular-sensibility.

All these conditions concurred in this case, and yet it was one of organic lesion of a serious kind.

The case affords a striking proof of the insidious origin and course of chronic spinal meningitis. The local action appears never to have been acute, and was unattended by those symptoms of irritation which are supposed to characterise inflammation of the membranes of the cord.

The changes in the cord were probably subsequent to those in the membranes and posterior roots of the nerves.

Cases xxx and xxxi are good examples of malignant disease about the spine affecting the cord. In one, the substance of the cord was sloughing; in the other, the proximity of the cancerous growth had induced only softening. In neither, was the nervous substance the seat of the new growth. At an early period, when there is nothing tangible, the symptoms in such cases are commonly referred to neuralgia; the word 'neuralgic,' for explaining symptoms, as the word 'idiopathic,' for explaining causes, being of so easy use that it invites careless investigation. But, for this, there are generally circumstances which would suffice for a sound diagnosis. The pain is more or less characteristic in its continuance and severity. Its seat about the spine is also a sufficient cause of suspicion, since this region is not commonly affected with pure neuralgia; added to which, collateral symptoms, if sought for, are often found to remove the difficulty. For instance, signs of pressure on the bronchi, where the dorsal region is the seat of the disease, or, as in Case xxx, the invasion of an adjacent organ by the malignant growth.

Case xxxii is one of strumous tubercle developed towards the centre of the cord. The chief value of the case lies in its history, for the patient being an infant, as the arms only were at first affected, the paralysis might not have been regarded as of serious importance. Young infants are occasionally the subjects of paralysis of one or both arms, from the carelessness of nurses in tying the dress so as to produce pressure on the axillary plexus. Not unfrequently also the paralysis of the period of dentition, the 'paralysie essentielle' of French authors, shows itself in one or both arms, whilst the legs

remain unaffected. In both these forms the onset of the paralysis is sudden, and by that alone they would be distinguishable from such a case as the above.

CASE XVII.—*Complete paraplegia without loss of sensation ; onset of symptoms sudden ; (acute tabes dorsalis.) Death after fourteen days from acute peritonitis set up by inflammation of the bladder ; no discoverable change in the structure of the cord beyond slight softening of the texture ; no exudation.*

(Reported by Mr. DURHAM.)

Henry P—, æt. 32, clerk to a solicitor in the city, was admitted under my care into Guy's Hospital, 23d December, 1857. A tall, well-made, rather pallid, but otherwise healthy-looking man, suffering from entire paraplegia of the lower extremities and sphincters, but without affection of sensation. He stated that he had never previously had any serious illness, but that two years ago he fell whilst attempting to jump over some chairs. After a few days, all apparent effects of this accident passed away, and he considered himself in unimpaired health. In the summer of 1857, he married, and gave himself to excessive indulgence in sexual intercourse. He was otherwise temperate. For two or three months preceding the sudden development of the paraplegia, he experienced at times some difficulty in micturition. The urethra was healthy. On the 9th of December there was numbness of the lower extremities extending as high as the knees, but this was so slight as not to attract any attention at the time. On Monday the 14th, he walked as usual from the suburbs to his business in the city. About the middle of the day, as he was crossing his room, his legs suddenly became weak, and he would have fallen had he not been supported. After a short time, he recovered sufficiently to walk with some difficulty to the omnibus, and afterwards from the omnibus to his home. In the course of the afternoon he became entirely paraplegic, the urine and fæces passing involuntarily from him. There was no affection of the upper extremities except slight and transient formication in the hands.

On admission, on the 23d, there was only a trace of excito-motor activity in the left leg, and none in the right. There was no appreciable diminution of sensation. Movements in the chest normal. Pulse 120, feeble. Pupils dilated. Surface of trunk and upper extremities warm and perspiring. Legs cold. A sense of tightness around the chest, about the attachment of the diaphragm. Bowels inactive. Urine drawn off by catheter, acid.

The day following his admission, there was noticed to be some œdema of the integuments in the lumbar region, especially on the right side. On the 26th, this had almost disappeared. The spine was normal. No change in the paralytic symptoms. Occasional slight involuntary twitchings of the legs. *Electro-contraction of the muscles good.* Only the slightest trace of excito-motor action and that limited to the left leg. The integuments over the sacrum reddened. Pulse 130. Skin hot and dry. Urine ammoniacal, and containing a large quantity of very offensive mucoid pus. The passage of the catheter was followed by much bleeding. During the night of the 28th nausea and vomiting came on, with great prostration. Respiration

thoracic. Death from exhaustion on the morning of the 30th, the case having been brought to a rapid termination by the supervention of acute peritonitis upon inflammation of the bladder. The upper extremities were unaffected throughout with the exception of the slight and transient formication noticed above.

Post-mortem examination.—Head not examined. About the base of both lungs, commencing acute lobular pneumonia. Lung-tissue otherwise healthy. Heart healthy. Intestines covered by recent inflammatory exudation. Mucous membrane of bladder sloughing. Its muscular coat, and the pelvic areolar tissue, infiltrated with fetid pus and urine. Two false passages, one passing through the prostate and thence into the bladder, and the other passing into the areolar tissue behind it. No stricture of the urethra. Texture of kidneys healthy. No trace of old or chronic disease could be discovered, either about the pelvis, in the pelvic viscera, or in the bodies of the vertebræ. The larger veins were opened, but afforded no evidence of phlebitis. Integuments over the sacrum beginning to slough, over the lumbar region they were œdematous. Membranes of the cord healthy. As the finger was passed lightly along the body of the cord it appeared to be somewhat softened at two points, in the middle, and at the lower part of the dorsal region; but on the most careful microscopical observation nothing abnormal was discovered in the texture either at these parts or in any other, though the cord was submitted to repeated and searching examination by the microscope. The epithelium lining the ventricle of the cord in the lower dorsal and lumbar regions was abundant, but normal. A few granules of brain-sand were found in the posterior columns, about the middle of the dorsal region. No traces of inflammatory exudation anywhere, either in the cord or in its membranes, nor any evidence of degeneration of the nerve-tubules.

Remarks.—When this patient came under care it was thought that the paraplegia was the result of ramollissement of the substance of the cord, which had (as not unusually happens) been more or less latent in its progress, the sudden paraplegia coming on when the conducting tubules have reached a point of degeneration which destroys their continuity. The examination of the cord did not confirm this diagnosis. In

the present state of nervous pathology, the case remains unexplained. It is confessedly difficult to establish a negative, but the difficulty was met with unusual care in this case. Hours were spent in the examination of the cord, but with no other result than to show that there was no appreciable lesion of it, besides a slight and doubtful softness of the tissue at two points. We may, therefore, certainly conclude that the spinal cord may have its functions impaired and even lost, and that suddenly, as far as the power of motion is concerned, without any distinct amount of anatomical lesion. Some writers have thought that the cord might be paralysed by a morbid impression made upon it, through incident nerves, and independently of any lesion of structure. Mr. Stanley sought to establish this in reference to disease of the kidneys, believing that these organs when congested, might, through their nervous connections, set up paraplegia. I have shown in another place (*'Med.-Chir. Trans.,'* 1854), that the cases recorded by that author do not support his theory. In the instance before us, there was no lesion of the kidneys or of the pelvic viscera, preceding the paraplegia; nor does there in the history of the case appear to be any sufficient cause for the paralysis, unless we accept it as one of acute *tabes dorsalis*, resulting from over sexual indulgence. Had the case not been rapidly terminated by cystitis and acute peritonitis, the cord, examined at a more advanced period, would, in all probability, have presented definite degenerative changes; or, perhaps, it might have recovered itself by the slow processes of nutrition. It is worthy of notice, that sensation was not affected. In the treatment, cupping, blisters, and mercury, would have been obviously inappropriate. Wine and opium moderately in the beginning; and at a later stage the mineral tonics, were the means indicated; but, unfortunately, as too often happens, the accidents of the paraplegia, the pelvic complications, gave no opportunity for the successful issue of the case. In this respect, women have the advantage over men, catheterism being less needed, or when required, less liable to produce injury in them than in the male sex.

CASE XVIII (Plates I, II).—*Numbness and weakness of legs for several months ; sudden onset of pain and increased debility ; no impairment of sensation ; temporary increase of voluntary power under the use of strychnia, soon followed by complete paraplegia ; retro-peritoneal abscess between bladder and uterus. Death from peritonitis : remarkable atrophy of the gray substance of the cord ; chronic inflammatory degeneration of the posterior columns.*

(Reported by Mr. DURHAM.)

Harriet B—, æt. 50, (?) but looking much older. A widow employed as a nurse. Admitted into the hospital 10th December, 1857. Seven weeks before this, she was suddenly seized with acute pain in the right foot, so severe that she could not move the leg. In a few days the left foot was similarly affected. The pain gradually subsided, but only to return at intervals as severely as ever. The muscular power became at the same time impaired. She could move the limbs when lying down, but not leave her bed. On questioning her, it appeared she had for some time felt uncomfortable sensations in the legs, with slight numbness, and a feeling of debility, but was able to perform her duties until the time of the sudden seizure of pain. When she came into the hospital, there was only just sufficient voluntary power over the legs to flex them slightly, the left rather the most. Occasional feeble involuntary jactitations, and distinct but not very marked excito-motor movements. Sensation not impaired. Urine drawn off by catheter, ammoniacal and containing mucus. Pain over the abdomen; occasional vomiting. Strychnia was given in doses of 1-16 grain. Under its use the voluntary power was for a few days rapidly increased, but at the end of a week the spasms of the legs were so violent the medicine could not be continued. The cord was left exhausted, and, at the end of five weeks after her admission, the legs were completely paralysed, and no excito-motor movements could be produced. Sensation now seemed to be impaired, but the patient at this period of her illness lay for the most part in such a dull and stupid state, that it was difficult to form a satisfactory opinion on certain points. The skin was abraded over the sacrum and trochanters. Highly offensive urine dribbled from the bladder. She would not submit to have anything done with the catheter, on account of the pain it caused her. She lay in a state of semi-coma, and died exhausted, February 5th, 1858.

Post-mortem examination.—Body wasted. Head not examined. Spinal bones, ligaments, and theca-vertebralis, healthy. Arachnoid normal, with one or two fibroid plates on the visceral layer. Spinal cord in the lower dorsal region small, and soft to the touch ; the anterior fissure gaped open. Examined in the fresh state, abundant granule-masses (exudation-cells, &c.), having the usual appearance, were found in the columns. On section, the centre of the cord formed an

irregular depression from atrophy of the gray substance. These changes were, however, more definite after the cord was hardened by immersion in spirit and thin sections made of it. The atrophy affected the fibrous portion of the gray substance. The caudate vesicles had their normal position and structure. There was no exudation amongst the gray substance. The symmetry of the changes in the columns, and the mode of extension of chronic disease in them, are well shown in Plates I and II. The lesion was due to chronic inflammation and concomitant atrophy of the tissue, with subsequent fatty degeneration of the newly effused matter. The fatty incrustation of the capillaries was a mechanical result, as shown in Case XXI. Cortical portions of both kidneys full of points of suppuration. Pelves, ureters, and bladder acutely inflamed. A large retro-peritoneal abscess between the bladder and uterus, but not communicating with the bladder. Recent inflammatory effusion over several coils of intestines in the pelvis. Viscera of chest healthy.

Remarks.—An inspection of the sections given in Plates I and II will show how much could be expected from treatment. It cannot be objected that the lesion there depicted does not convey a true impression of what existed during life, since there is no evidence of recent changes. It is an important consideration in the treatment of diseases of the nervous centres, how far the symptoms are due to irremediable changes or not. A lesion of the nervous tissue may be cured—or, at least, be in a state which, if it were in the skin, or muscle, or gland, would be called cured—and yet, according to the patient's estimation, the disease may remain. We too often think of symptoms as substantially the disease; and if this false view guides our treatment we cannot fail of doing harm. We waste the feeble powers of an already partially dilapidated system, instead of recognising the dilapidation as an essential and permanent condition of the body we have to treat.

The therapeutical agency of strychnia in organic lesions of the cord has yet to be proved. Judging from its effects, we should say its direct operation on the tissue was the very reverse of nutritive or reparative. If function is, as there can be no doubt, the effect of a mode of disintegration, agents

which directly increase function must produce a disintegrating action. If this be a sound inference—and experience leads to the same conclusion—strychnia has but a limited therapeutic application in paraplegic affections. It is well known that immediate and striking effects can be produced by this drug, but these are often followed by hopeless bankruptcy of the spinal power. In giving strychnia, our object should be to produce no greater change of the tissue than shall, by the stimulus of waste, increase the power of nutrition, as we exercise an organ to favour its healthier growth. This requires not only a diagnosis of the conditions producing the paraplegia, but a careful adaptation of the dose of the medicine, which is often a difficult point. I have seen one twenty-fourth of a grain given twice a day for only two or three days, in a case of chronic paraplegia, apparently depending upon softening of the cord, set up very decided irritation. So unstable is the structure of the nervous tissue in some of these cases, and so delicate, in proportion, must be our interference by remedial agents.

CASE XIX (Plate III, A, B).—*Chronic inflammatory degeneration of the posterior columns of the cord throughout their whole length. The disease strictly limited to the posterior columns. Frequent vomiting. Emaciation of the voluntary muscles generally. Paraplegic weakness of lower extremities, characterised by a want of control over the contraction of the muscles. Congenital misplacement of the ascending colon, which became twisted on itself. Cæcum sloughing.*

William J—, æt. 28, of the middle stature; fair hair; emaciated; anxious expression; large head; broad and prominent forehead. Though he had never been robust, he had good health until the beginning of the year 1857. He was first seized with vomiting, which came on without any discoverable cause, and lasted for several days. As he recovered from the attack the legs became weak. After three months he had a second attack of vomiting, followed by an increase of weakness in the legs. He was admitted into Guy's Hospital under my care, November 11th, 1857. He was then unable to stand without support. In a recumbent position he could flex and extend the legs with some freedom, but the movements were sudden and vague, from want of control over the action of the muscles; the spinal centres, when stimulated by the will, seeming to shoot off their influence at once, making the feeble muscles contract to their full extent with a jerk. In other words, there was no power to regulate the muscular contraction. The movements of the fingers

were also wanting in precision. He was awkward in handling small objects, or in applying the hand to grasp larger ones. The muscles were thin and flaccid, corresponding to the general emaciation of the body. The muscular irritability was excessive. Weak currents of electricity, not sufficient to affect healthy muscles, excited well-marked contraction; whilst a little stronger, but yet very moderate dose of electro-magnetism, produced cramps lasting for several seconds after the stimulus was discontinued. The arms were weak, with an obvious want of control over the voluntary movements. There was numbness of the feet and hands, and a burning formication in the fingers and toes. The sensation of the other parts of the body was normal. No involuntary contraction of the legs. Sphincters good. Urine acid. The lower ribs depressed, and but little moved in inspiration. Headache; vertigo; cerebral confusion; tinnitus. Pupils largely dilated, the left the most so; sight dim; occasionally transient amaurosis. Sleep disturbed by dreams. Frequent nausea and vomiting, with pain from the epigastrium to the spine. Abdomen not distended; soft. Skin hot and perspiring. Pulse permanently quick, 126, small, and feeble. Respiration 32. Spine straight. No tenderness on pressure or percussion. He could give no account of any accident or injury to the spine, except such as might have resulted from a fall, flat on the back, from the height of a few feet, eight years before his symptoms began. His habits had been temperate. No syphilitic taint.

After admission into the hospital he continued to have repeated attacks of vomiting, lasting for many days, uninfluenced by any remedies. The vomited matters were copious, greenish, and mucous. The bowels continued to act freely, but without relief to the sickness. The irritability of the stomach was attributed to the state of the cord. The attacks of vomiting increased his anxious aspect. The paraplegic symptoms continued unchanged. There was, as before noted, headache and vertigo, and sometimes transient amaurosis. The pupils remained permanently dilated, and with the same inequality. The pulse quick (120 to 130), and feeble. On only one occasion was the urine noticed to be alkaline, when first passed. It never contained mucus. There was no band-like sensation around the abdomen. He often complained of pain from the epigastrium through to the spine. About the middle of February, 1858, he first had a sense of bearing down about the rectum, and complained of great distress after an action of the bowels, and of startings in the legs. March 8th he had an attack of vomiting, apparently such as had often occurred before. This continued on the 9th. On the 10th he was collapsed and pulseless, with cold sweats, and other symptoms of ruptured intestine. There was no cerebral oppression. He died on the 11th.

Post-mortem examination.—Body emaciated. Brain healthy. Thoracic viscera healthy. Fecal extravasation into peritoneal cavity. Intestines adherent by recent lymph. Omentum contracted into a cord-like mass, and firmly adherent to the left side over the pubis. Cæcum fallen into the cavity of the pelvis. From it the ascending colon passed directly to the left side towards the spleen, and then curved down again before becoming continuous with the descending colon. This displacement of the cæcum and ascending colon arose from a

congenital (?) absence of the meso-colon on the right side. The ascending colon, at its commencement, was partially twisted upon itself. Both it and the cæcum were dark coloured, and sloughing to a large extent, from mechanical obstruction. The spinal cord had its normal appearance and consistence, except, perhaps, a small portion in the dorsal region, which seemed rather softened; but this was doubtful, and was only such as an accidental tension in moving it from the canal might have produced. Sections of the cord made at the time gave no further evidence of disease. The membranes were healthy. After hardening the cord, and making fine sections, it was seen that the posterior columns were atrophied throughout their whole length, and amongst the tissue were numerous exudation-cells in a state of fatty degeneration (granule-cells). The posterior roots and the lateral columns were normal (Plate III). The disease was limited above by the commencement of the medulla oblongata. No degeneration of structure had occurred in this part.

Remarks.—The relation of morbid conditions to each other is often difficult to determine. It was so in this case. The oldest disease was no doubt that discovered in the abdomen; the absence of the meso-colon on the right side was evidently congenital, and probably the adhesion of the great omentum dated also from an early period. It was these lesions which brought about the fatal result. The steps of the process may be looked at in two ways. It may be admitted that a cæcum and colon left to float free were in danger of getting into positions unfavorable to the propulsion of their contents, and thus of occasioning attacks of vomiting, such as ushered in the other symptoms in this case, and continued to harass and distress the patient throughout his illness. It may also be thought probable that attacks of abdominal disturbance might, through incident nerves, set up a secondary lesion in the cord. This, however, is doubtful, and it is more in accordance with our pathological views to refer the early attacks of vomiting to the lesion of the cord itself, as the primary disturbance, especially since the degeneration of the columns extended up to the neighbourhood of the medulla oblongata. With this view, it is not difficult to understand

how the congenital defect in the colon should be brought into fatal operation by irregular peristaltic action so induced.

The limitation of the disease to the posterior columns was remarkable (Plate III, A, B). Though they were degenerated throughout their whole length from the lumbar portion to the medulla oblongata, neither the posterior roots of the nerves, nor the adjacent parts of the lateral columns, were in any way involved in the degeneration. We might, from this strict limitation of the lesion, hope to gain some unequivocal evidence as to the physiology of these structures.

The affection of sensation was limited to numbness, and formication of the hands and feet. Dr. Brown-Séquard has shown, by transverse section of the posterior columns in animals, and by instances of disease in the human subject, that where the posterior columns are destroyed for a limited extent, as by pressure of a tumour, hyperæsthesia is produced in the parts below the injury; in both extremities if the lesion affects both columns, but only on the side of the lesion if one column is affected. When, however, the posterior columns are destroyed throughout their whole length, instead of hyperæsthesia, there is loss of sensibility to some degree. Not that the posterior columns convey, according to this observer, sensitive impressions to the brain, but, because, being in part channels through which the fibres of the posterior roots reach the gray matter, if they are destroyed or degenerated throughout, a certain number of sensitive fibres must be destroyed also. So far theory coincides with the facts noticed in this case.

The same physiologist believes that the special function of the posterior columns is for the reflex movements. These functions ought, therefore, to have been destroyed, or at least greatly diminished. Nothing in favour of such a theory was, however, noticed, except the general muscular emaciation.

The sphincters of the rectum and bladder continued to perform their office. The muscles remained in a state of hyper-excitability to the galvanic stimulus. I do not know how far the state of the colon might be referrible to a loss of the reflex power.

Neither were the phenomena more in favour of the value of the test proposed by Dr. Marshall Hall, for the diagnosis

of cerebral paralysis from spinal paralysis, since according to that, the irritability of the muscles should have been much diminished, the disease being in the cord; but, on the contrary, it was remarkably increased.

This brings us to the theory of the posterior columns proposed by Dr. Todd, that they "propagate the influence of that part of the encephalon which combines with the nerves of volition to regulate the locomotive powers, and serve as commissures in harmonising the actions of the several segments of the cord." The want of power in this case to regulate the action of the muscles was very characteristic. The legs, when drawn up, as they could be freely, were drawn up with a sudden jerk, and extended in the same manner. The voluntary movements of the hands were also fumbling and vague.

The limitation of the disease to the posterior columns, coincides with what is generally found. There is evidently a tendency in lesions to spread longitudinally in the cord rather than transversely through it. Probably from homogeneity of structure or from the arrangement of the blood-vessels.

Such complete and symmetrical isolation of a structure is very suggestive of an independent function.

It is unnecessary to refer particularly to the character of the morbid changes in this case. They were evidently of that kind which we denominate by the term "chronic inflammation." Atrophy of the proper tissue, with exudation, which corpusculates and then becomes fatty. This change was probably induced by the fall on the back eight years previously.

In the ordinary mode of examination the disease of the cord in this case must have been overlooked, and it would probably have been regarded as one due to cerebral disease, though certainly there was no evidence of it post mortem. Clinically, there was more to support such a view, namely, headache, vertigo, cerebral confusion, tinnitus, dilated pupils (one larger than the other), dim vision, occasionally transient amaurosis, sleep disturbed by dreams, &c. These symptoms—together with power to move the limbs when in a recumbent posture, but inability to stand without support, and apparently a great increase of all the symptoms when the patient is in a

vertical position—led Dr. Baillie to assume that the seat of the disease in these cases is in the encephalon. In the year 1848 I proposed a classification of paraplegia which should recognise the existence of such cases; but a better method of investigating the morbid changes in the cord daily lessens the number of instances referrible to such a division, and makes it doubtful whether paraplegia properly so called is ever due to lesions which are strictly cerebral in their seat.

CASE XX.—Paralysis of both seventh nerves; nearly complete paraplegia of lower extremities; weakness of upper extremities; onset of symptoms acute. Death on the ninth day. For some months preceding the invasion of the paraplegic symptoms pains in the left arm and slight wasting of the muscles, supposed to be rheumatic; wasting of the gray commissure on the left side of the cord in the cervical region; recent inflammatory exudation into the tissue of the medulla oblongata and into the gray commissure of the cord.

Mr. E—, æt. 59, began to suffer from pain in the left arm, from the shoulder to the elbow, at the end of the year 1856. The pain continued some months, and left the arm weak and slightly wasted. This was regarded as a rheumatic affection. There was no anæsthesia; Mr. E— was in other respects in good health until Christmas, 1858. He could give no more precise description of the beginning of his indisposition than that he was languid. He spoke also of an occasional feeling of coldness between the shoulders and down the spine, attended with distressing rigors in the spinal muscles. At the beginning of March, 1858, he had bilious vomiting, with pain in the right hypochondrium. He was able to continue the active duties of his profession as a medical practitioner, and gave a public lecture on the evening of the 29th of March, but said that he felt more weak and tremulous on that day than usual. He visited his patients on the 30th, and appeared in his ordinary health, but in the evening complained of constricting pains in both arms, from the shoulders to the insertion of the deltoid muscles. On rising from bed on the morning of the 31st he found his legs too weak to support him, and from that time his paraplegic symptoms rapidly increased. I visited him on the 4th of April. There was then entire inability to move the muscles of expression on either side of the face. The involuntary action of the orbiculares palpebrarum continued, but the eyes could be only partially closed by volition. The features hung motionless. He first noticed the paralysis of the face the day previous, when attempting to put up his lips to kiss his wife. The motor and sensitive divisions of the fifth nerves were unaffected, except slight anæsthesia of the first division on the left side. Motions of the eyes, vision, hearing, taste, and deglutition normal. Respiration chiefly abdominal, the movements of the lower ribs being defective. Numbness of the fingers

of both hands. Movements of upper extremities free. He lay supine with the legs extended and powerless. Muscles flaccid. No excito-motor movements on irritating the soles of the feet; sensation impaired, and a feeling as of a board pressed against them. The sphincters retained their power. Urine pale straw-colour, acid. Intellect perfectly clear. Tongue protruded straight. Articulation perfect for all words not requiring the use of the lips. Labials could not be pronounced. Pulse 72. Respiration tranquil. On closer inquiry as to any premonitory symptoms it was elicited that, in the summer of 1857, on one occasion, in sleep, an evacuation had passed from him involuntarily. This was the only evidence of spinal disorder, except that given above. On the 6th there was a slight return of power over the muscles of expression. The paraplegic symptoms, however, were unchanged. On the 8th the respiration was more feeble, and chiefly abdominal. Tongue dry and brown. Dribbling of urine. When roused he was quite collected, but left to himself there was wandering delirium. Movements of arms very feeble; slight subsultus. He died in the evening very tranquilly, the breathing ceasing so gradually that the last respiration could not be told.

Post-mortem examination.—Rigor mortis well marked, both in upper and lower extremities. Large amount of subcutaneous fat over chest and abdomen. Muscles of lower extremities well developed. Left arm slightly less muscular than right. Large deposit of fat about the base of the heart and over the right ventricle; valves healthy; aorta extensively atheromatous. Lungs healthy. Universal, old, tough adhesions between the diaphragm and upper surface of liver. No corresponding adhesion of the pleura above. Kidneys large, tunics easily stripped off; surface smooth. Bladder healthy. The convolutions of the hemispheres of the brain separated by clear sub-arachnoid effusion. No other abnormal change. Corpora striata, thalami optici, cerebellum, and pons varolii healthy. The basilar and vertebral arteries opaque and rigid. The trunks of all the cerebral nerves healthy. Bones, ligaments, and membranes of the spine healthy. In the cervical and dorsal regions the substance of the cord was to the touch somewhat softer than natural, but no other unequivocal change was discoverable by the unassisted eye or by the aid of a common lens. On hardening the pons varolii, medulla oblongata, and cord, and preparing sections after a modification of Lockhardt Clarke's method, it was seen that in the anterior part of the commissure, throughout the length of the cord, but principally in the lumbar and superior cervical regions, and throughout the structure of the medulla oblongata, but chiefly at its superior part, there were exu-

dation-cells scattered interstitially amongst the tissue; they were also seen, but more sparingly, in the lines of areolar tissue which radiate through the white substance and amongst the deeper part of that which dips into the anterior commissure. There was no want of continuity nor any destruction of the nervous tissue. The cells were recent. They had not undergone fatty degeneration. The amount of the exudation was so small and its distribution such that no lesion was visible, except under the higher powers of the microscope. It was then very distinct, and remains so in the sections preserved in Canada balsam. In the cervical region on the left side there was wasting of the gray commissure and a development of fibrous tissue in its place.

Remarks.—The supposed rheumatic affection of the left arm was referrible to the changes in the gray commissure in the cervical region. This change was very limited in extent, but still very definite when transparent sections of the cord were examined. The experiments of Brown-Séguard—which go to prove that injury to the gray matter of the cord on one side alters the sensibility on the opposite side of the body—seem opposed to the facts in this case. It is probable, however, that the painful affection of the left arm was due to a lesion of the motor nerves—the chronic changes in the muscles subjecting the textures to unnatural tension.

The paralysis of both seventh nerves was a striking incident in the case. It was referrible to the central changes which extended through the tissue of the medulla oblongata. The nerve-trunks and surrounding parts were healthy. The exudation estimated in mass was very trifling, not sufficient indeed to give unequivocal evidence of its presence but for our improved methods of research. It is not, however, to be forgotten that its seat was in the most important part of the nervous centres, where nature has afforded no surplusage.

The defective speech led those about the patient to suppose the symptoms were due to disease of the brain. It, however, needed but little investigation to show that this defect was entirely due to paralysis of the lips, and was limited to the pronunciation of labials, other parts of speech being pronounced distinctly.

The intellect was undisturbed. The patient gave a very clear

account of himself. The tongue was moved freely. Digestion unimpaired, and the breathing natural.

The diagnosis was of softening of the cord, but strictly speaking this was not the lesion. It was an inflammatory exudation into the more vascular parts of the cord and medulla oblongata. To what condition of the circulating fluids or of the blood-vessels this was attributable is conjectural. The patient was a beer- and porter-drinker, his subcutaneous tissues were loaded with fat, his age 59:—conditions which are associated with and favour a gouty state, no doubt much oftener than the occurrence of distinct gout would seem to indicate, for a patient may be gouty who has never had gout, as one may be poisoned by marsh miasm who has never had ague. There was no history of injury or of exposure to cold. The effusion under the cerebral arachnoid was probably the result of that capillary paralysis (congestion) which comes on in death from disease of the nervous centres.

CASE XXI.—Sudden paralysis of right leg; partial recovery, after five months, acute paraplegia; erysipelas and consecutive pneumonia from the application of heat to the legs. Irritative fever, and death in two weeks. Recent softening of the cord in the dorsal region; old degeneration of the right lateral column, with fatty incrustation of the capillaries.

Ed. M.—, æt. 34, a man of dissolute habits, but originally of a strong and well-developed constitution. At the end of November, 1853, on rising from his bed felt himself suddenly powerless in the right leg. He had at the same time pain in the lumbar region, extending to the hypochondria. There was no anæsthesia. He asserted that he had felt nothing wrong with himself previously; and, so far as he knew, there had been no premonitory symptoms of the paralysis. For more than a month the leg remained completely paralysed, “there was not the slightest power of motion in it.” Pain in the course of the sciatic nerve. Urine drawn off by the catheter; ammoniacal. Frequent priapism with spermatic discharges (as proved by microscopic examination), but he was not himself aware of any excitement of the genital organs when questioned about it. He was treated by Mr. William Hills with laxatives, the preparations of iron and strychnia, and by galvanism. Slowly the pain left him, and he recovered some power over the leg so as to be able to stand upon it, and to walk with the aid of a stick, but he could not flex the muscles of the hip-joint. With this improvement, he relapsed into his former habits, and after a week rather suddenly became paraplegic. He was admitted into the hospital April 29th, 1854. The right leg was then wasted and completely para-

lysed. Slight power remained in the left leg, and there was frequent involuntary jactitation of it. It was swollen from erysipelatous inflammation which had extended from a bulla caused by the application of a hot bottle. Irritative fever followed, and death at the end of a fortnight from the time of the relapse.

Post-mortem examination.—On the left leg superficial excoriations and the remains of blebs. Subcutaneous cellular tissue containing collections of pus. Saphena vein not implicated. Bones, ligaments, and membranes of spinal cord healthy. Large quantity of transparent cerebro-spinal fluid. In the upper dorsal region the substance of the cord was softened to the extent of an inch; exudation-cells scattered through the tissue. In the right lateral column, near the same part, the tissue was atrophied and the capillaries incrustated with oil-globules (fig. 1).

Fig. 1.¹



The fatty matter could be removed by ether, leaving the walls of the vessels apparently normal. This change was strictly limited to the right side of the cord. Recent lymph on lower lobe of right lung. Pneumonic consolidation of the bases

¹ Sketch of capillaries incrustated with oil-globules, case XXI.

and posterior parts of both lungs. Liver pale, weight four pounds three ounces. Kidneys congested, weight 12 ounces. Mucous membrane of bladder thickened, congested, and greenish.

Remarks.—The chief pathological interest of this case lies in the suddenness of the paralysis in the first instance, and its limitation to the right leg. The cause of this was plainly made out on examination of the cord. The incrustation of the capillaries with oil-globules appeared to be nothing more than a mechanical result, and not due to a degeneration of the coats of the vessels. In pathological changes of the nervous substance we may distinguish these two conditions. In the one, the changes in the capillaries are probably antecedent to the lesion of the textures, and in the other consecutive to it.

It is a matter of speculation what set up the softening. Dissolute habits induce many conditions predisposing to such a change; and amongst them, perhaps, none more efficient than the contamination of the syphilitic virus. In softening of the brain there can be no doubt of this connexion, and that, too, apart from any noticeable cachexia.

The seat of the softening corresponded with that so frequently found in other cases. The dorsal region, from its position and organization, is exposed to lesion, and the cord has, perhaps, at this part, less resisting power than at other parts which are more highly organized.

CASE XXII.—Paraplegia supervening two days after a violent exertion in lifting a heavy weight; softening of the cord opposite the fifth and sixth dorsal vertebræ; no injury of the membranes, ligaments, or bones of the spine. Death after six weeks.

Richard A.—, æt. 25, of a rather delicate constitution, was at his usual occupation as a labourer in the Commercial Docks, on Saturday, November 22d, 1856, when, after lifting some deals, he felt a sudden pain in the back. He walked to his home, the distance of a mile and a half, and the following day was apparently quite well. The next morning (Monday), on waking, the legs were paralysed. When admitted into the hospital, November 26th, there was complete paraplegia, a bed-sore had already begun to form over the sacrum, and ammoniacal urine dribbled from the bladder. He died exhausted, January 2d, 1857.

Post-mortem examination.—Body emaciated ; large bed-sore exposing the whole length of the sacrum. The bones and ligaments of the spine in the other regions were carefully examined, but no trace of injury was discovered. Opposite the fifth and sixth dorsal vertebræ the cord was softened through all the columns into a thick, greenish, muco-puriform fluid, with a tinge of brown. Examined by the microscope, it was seen to consist of disintegrated nerve-tissue, with a few irregular collections of granules. *The cord was not enlarged at the softened part, nor was there any trace of inflammatory exudation in it or upon the membranes covering it*, though to the unassisted eye it had the appearance of an irregular undefined abscess. The lumbar and cervical portions of the cord had the normal appearance and firmness. A large portion of the lower lobe of the right lung and half the upper lobe of the left were hepatized. Heart normal. Liver large and fatty. Commencing suppuration in the cortical substance of the kidneys. Mucous membrane of the pelves greenish, with patches of adherent fibrinous exudation. This condition of the mucous membrane was continued through the ureters into the bladder. The bladder contained a quantity of muco-purulent fluid.

Remarks.—This case shows that the substance of the cord may receive an injury through violent muscular exertion, whilst the surrounding textures escape. Why this should rather occur in the dorsal region is obvious ; since the curve of the column is most marked and most variable, and the body of the cord is thinnest, at this part. It is a matter also of common clinical experience that the cord is very prone to softening in the dorsal region, from which we may, perhaps, infer that, in addition to its being here more subject to injury, it has a more feeble organization than the cervical and lumbar enlargements. The change in the cord was seen by the microscope to be due to mere disintegration. There was no evidence of any plastic exudation. The greenish and brownish tints of the softened part were probably due to blood-colouring matter. We may infer, from the quality of the local changes, which appear to have been quite passive, and from this slight coloration, that the immediate effect of the injury was upon the capillary circulation, leading to effusion of blood and consequent atrophy.

CASE XXIII.—*Concussion of the cord in the cervical region from direct violence; ecchymosis into posterior horn of gray matter on left side, also into anterior horn on right side and into the posterior columns; loss of sensation immediately after the accident, followed by hyperæsthesia; paralysis of legs, left arm, and sphincters. Death thirty-four hours from the accident.*

(Reported by Mr. BANKART.)

Joseph K—, æt. 33, a coal-porter, strong and healthy, was carrying a sack of coals on his back, down some cellar-stairs, when his foot slipped forwards from under him and he fell, the sack of coals falling upon him. On his admission, immediately after the accident, 3 p.m., June 22d, 1858, there was loss of motion of both legs and of the left arm. The sphincters were paralysed. There was entire loss of sensation in the left arm as high as the deltoid. The right arm he could move, and had perfect sensation in it. On examining the state of sensation in the lower extremities, it was found that he could feel about the feet and on the outer side of thighs, but not on the anterior and inner surface. During the time marks were being made on the skin to indicate the state of the sensation at different parts, it was found to vary, returning to spots where it had just previously been absent. Apparently the most distant parts recovered first. Slight priapism. Breathing diaphragmatic. After a few hours, sensation returned in every part. As the skin became warm, he complained of pain when lightly touched. For instance, when the finger-nail was passed but lightly along the skin he would exclaim, "Don't prick me; don't hurt me!" The day following, the sensibility of the surface appeared to be excessive, judging by his exclamations when the skin was touched or pinched. This was especially noticed in the right arm. Priapism, which existed when he was admitted, passed off after two hours, but returned the day following. He continued to have power to move the right arm. He died thirty-four hours from the accident.

Post-mortem examination.—The spine only was examined. There was no external trace of the injury; no displacement of the vertebræ discoverable by external examination. The membranes of the cord were healthy. Opposite the fourth and fifth cervical vertebræ the substance of the cord was contused. On section, there was found ecchymosis of the posterior horn of gray matter on the left side, and of the adjacent part of the lateral and posterior columns. There were also other limited spots of ecchymosis on the right side, one in the right posterior column, and one in the anterior cornua of the gray substance. The gray matter generally was hyperæmic (from venous congestion?), but there was no other lesion of it, except

at the two spots named; no lesion of the anterior columns. The commissure was uninjured. On examining the spinal canal after the removal of the cord, nothing abnormal was discoverable in the bodies of the vertebræ opposite the lesion of the cord; but on dissecting off the posterior ligament it was seen that the body of the fourth was separated from that of the fifth, and that the left articular process of the fourth had been chipped off by the violent pressure of the lower one against it.

Remarks.—There are several points worthy of note in this case; the character of the injury received by the cord, namely, limited capillary ecchymosis; the absence of any external sign of the injury; the mode by which the cord was injured, namely, by concussion, and not by pressure of surrounding parts upon it, as shown by the ecchymosis being in the substance of the cord, whilst its peripheral parts and membranes had escaped; the limitation of the injury, producing paralysis of the left arm, whilst the right retained the power of motion; the immediate effects of the concussion on the cord, producing anæsthesia for a few hours; the return of sensibility first in the parts most distant from the injury, and the development of hyperæsthesia. This latter symptom was in accordance with the experiments of Séquard, who has shown that injury of the posterior cornua of the gray matter is followed by hyperæsthesia of parts below. Cases of injury, as before remarked, have as much interest to the physician as to the surgeon, since they often come under the care of the physician for the treatment of the permanent effects; when it is necessary there should be a correct estimate of the character of the primary lesion.

CASE XXIV.—*Concussion of the cord by a fall; recovery of power after two hours. Subsequent effusion of blood outside the theca vertebralis in the neck. Paraplegia of upper and lower extremities. Paralysis of intercostals. Intense heat of skin. Death in fifty-five hours.*

(Reported by Mr. VENOUR.)

Robert L—, æt. 40, fell backwards from a moderate height, a heavy plank falling at the same time upon him. He was at once brought to the hospital (4 p.m., July 7th, 1858). He was collapsed, but sensible. There was entire paralysis of the left leg, partial of the right, and also partial paralysis of the arms, but he was still able to flex the fingers. After two hours he had so far recovered from the immediate effects of the injury, that he could draw up his legs and grasp the hand; the circulation was improved; surface warmer. No injury of spine discoverable. At 10 p.m. he said he felt comfortable. He passed a restless night, and the following morning, at 8 a.m., was entirely paraplegic both in the upper and lower extremities. Loss of sensation in the paralysed parts. Priapism. Ribs scarcely moved in inspiration. Temperature of surface increased. Abdomen tense and tympanitic. During the day the skin became intensely hot, but the actual temperature was not noted. The breathing was wholly diaphragmatic. Deglutition difficult. He died fifty-five hours from the accident.

Post-mortem examination, by Mr. Bryant.—No external evidence of injury to the spine. On dividing the soft parts, there was found a separation between the fourth and fifth cervical spinous processes, and dislocation of the articular processes. The inter-spinous and capsular ligaments were torn through. Extravasation of blood outside the theca vertebralis on its anterior aspect. The effused blood compressed the cord, which was otherwise uninjured. After careful examination there were not found any signs of bruising of its tissue. The extravasation apparently arose from injury to the lower part of the body of the fourth vertebra, which had been fractured, and the inter-vertebral substance torn. The calibre of the canal was slightly encroached upon by displacement of the fourth vertebra, but not so as to press on the cord. The extravasation, though most abundant opposite the injury, extended downwards to some distance. The membranes of the cord were uninjured.



CASE XXV.—*Cervical paraplegia following an injury. Progressive muscular atrophy of the upper extremities, most marked on the side of the principal lesion in the cord. Anæsthesia, with severe neuralgic pains on the opposite side. Paroxysms of hiccup for several months. Thickening and adhesions of the membranes of the cord. Degeneration of the posterior columns. Dilatation of the ventricle of the cord. Opacity and fatty generation of the arachnoid of the brain. Ependyma of ventricles granular.*

John G—, æt. 49, a coal-waggoner, was forced backwards from his seat by striking his head against a beam, whilst driving under an archway. Several ribs were fractured on the left side. Some months after this accident he began to suffer pain from the occiput down over the shoulders, and in about a year the muscles of the upper extremities began to waste. After two years, incontinence of urine gradually came on. He was admitted into Guy's Hospital February 11th, 1857, three years from the time of the accident. He then presented a remarkable example of muscular atrophy without actual paralysis. The upper extremities were principally affected. The extensors of the right hand, the muscles of the thumb, and the interossei, were extremely wasted. The wrist dropped. The muscles of the shoulder and arm, including the pectoralis major and minor, much wasted, but in a marked degree less so than those of the forearm and hand. Very slight diminution of sensation. He could still lift the arm over the head. The left arm was similarly, but less affected than the right, so far as regards muscular atrophy, but there was numbness through the whole arm down to the fingers, and he suffered severely from neuralgic pains in it, which greatly depressed him, and which he described as a compound of smarting and numbness. The trapezii, serrati postici superiores, rhomboidei, and all the long muscles of the neck and back, were remarkably atrophied. The spinous processes were very prominent. No deformity nor tenderness on pressure at any point. The intercostals were so weak that the only respiratory movement was through the diaphragm. The supra spinati were atrophied, but not to the same extent as the infra spinati and levatores anguli scapulæ. The legs were wasted and weak, but he was able to walk. Sphincters weak. Dribbling of urine. Constipation. The thorax looked narrow and ill developed, from the wasting of the pectorals, the intercostals, and erectores spinæ muscles. The muscles at the back of the neck and the sternomastoids were so weak, that the head could not be supported erect. Sight dim, drooping of left eyelid. Frequent hiccup for many months. After his admission his principal complaint was of pain in the left arm from the clavicle to the fingers. He described it as a severe smarting, with a sense of numbness. His distress from this cause was very great. At the early part of March febrile symptoms set in. Tongue became dry and brown. Frequent hiccup and vomiting. Pain in left arm severe. Dyspnœa. Died March 25th, 1857.

Post-mortem examination.—The arachnoid of the brain

opalescent, with spots of white mottling of the more opaque parts from fatty degeneration. Subarachnoid fluid in excess. Ependyma of lateral and fourth ventricles granular, in the latter extremely so. The dura mater on the posterior surface of the cord much thickened. The two layers of arachnoid adherent in patches along this surface, and much thickened by effusion of lymph of old date. Sections of the cord examined with the naked eye gave no distinct evidence of disease. There was a slight yellowishness of the posterior columns, and increased vascularity and thickening of the pia mater covering them. In these columns, especially in the right one, abundance of granule-cells were discovered by the microscope. The exudation was greatest in the middle and lower third of the cervical enlargement. The gray matter was hyperæmic. No exudation into its tissue, nor into the anterior columns. The ventricle of the cord enlarged and distended with delicate granular nuclei. The affection of the cord appeared to be secondary to chronic inflammation of the membranes, and to chronic changes in the ependyma of the ventricle in common with the ependyma of the fourth and lateral ventricles of the brain. Hypostatic engorgement of both lungs, several lobules consolidated from recent pneumonia, some grayish. Other organs healthy.

CASE XXVI.—*Progressive atrophy of the muscles of the trunk and upper extremities, after a blow on the neck with the fist.*

Daniel C—, æt. 15, received a blow with the fist between the shoulders from a boy at play. After a week the head drooped, and gradually from that time the muscles of the upper extremities wasted, the arms dropped and hung useless, the intercostals lost their power, and the breathing was diaphragmatic; the lower two thirds of the trapezii and the erector spinæ muscles also wasted in the same way. This sketch was made fourteen months from the injury, to exhibit the wasted condition of the muscles and the position of the head and trunk; the head fallen forwards and the trunk thrown backwards to balance it, in the absence of muscular power.

The flattening of the ribs from the paralysis of the intercostals was such that the heart beat to the right of the left nipple and between the third and fourth ribs.

The patient was able to walk about when the sketch was taken. His gait was vacillating, but apparently more from want of muscular power to fix the trunk on the pelvis than from defective power in the legs. He could not sit on a seat without a support to the back. Sphincters good. On testing the electro-contractility of the

Fig. 2.



wasted muscles, by galvanism, they were found to contract in proportion to their mass; those muscles of the upper arm, which were the less wasted, contracted well; those of the forearm and hand, which were the more wasted, contracted less, but still distinctly. The progress of the disease was unattended with any pain. The wasted muscles not tender. No flickering contractions of their fibres.

Remarks.—This case is recorded as a good illustration of progressive muscular atrophy after concussion of the cord. It is to be observed, that there was no more paralysis than was due to atrophy of the muscles, and that the electro-contractility of the muscles was in proportion to their bulk. These facts are of importance, since it has been erroneously proposed to determine by the test of galvanism the diagnosis between progressive muscular atrophy from morbid changes primarily in the muscles, and that muscular wasting which is consecutive to disease of the cord. It is said that, in the latter case, the muscles early lose their electro-contractility, a statement at variance with extended clinical observation, and further illustrated in Case XIX. No doubt, as the lesion of the cord advances in this case (which is still under treatment), the lower extremities will undergo the same changes as the

Fig. 2. Sketch showing wasting of muscles after a blow on the neck.

upper. A precisely similar instance (Case xv, with post-mortem examination), was recorded in the 'Reports' for 1857.

CASE XXVII.—*Acute rheumatic (?) affection of the larger joints. Paraplegia of lower extremities. Slough over sacrum. Recovery.*

Anne E—, æt. 39, was admitted into Guy's Hospital, March 31st, 1857, under the care of my colleagues, Dr. Hughes and Dr. Wilks (to whom I am indebted for placing the case at my disposal). Both hands were swollen, stiff, and painful, with an erythematous blush over the back of the right, and on the second joint of the thumb of the left. The legs were so far paralysed that she could only very slowly and feebly move them. The muscles were greatly wasted and flabby, but had not lost their excitability by galvanism. Sphincters weak. No swelling of the knees or ankles at this time. Sensation nearly normal, but at times both legs felt numb, and were drawn up involuntarily. Urine acid, high coloured, and scanty. Tongue covered with a cream-like fur; skin hot, perspiration profuse, with acid smell. Pulse 120; systolic murmur over ventricle. On examining the spine, the lower third of the sacrum was found to be bent forward, the result of a fall eleven years before; and near the sacral notch, on the right side, was the cicatrix of a wound which formed at that time. Except this, there was nothing abnormal, nor any pain or tenderness on pressure. The history she gave of her case was, that being a widow, she was necessitated to work laboriously at a mangle. She had for two years, when much exerting herself, felt pain in the back between the shoulders, and a sense of constriction and coldness round the chest. Ten days before coming into the hospital she was seized with pain in the left leg, and had spasmodic contraction of the muscles, with an increase of the pain, and constriction round the chest. She had still power to extend the leg, but could not walk. The day following, the hands, knees, and ankles were swollen and painful. With these symptoms there was febrile heat and diarrhœa. The sphincter ani was so weak that the feces ran from her involuntarily. On the third day a slough formed over the sacrum. No important change occurred in her symptoms after her admission. There was great muscular emaciation generally. Involuntary twitchings of the muscles of the arms and legs. Aching, gnawing sensations in both calves. Touching the feet gave rise to formication, and very lively excito-motor movements. For ten days the hands remained red, painful, stiff, and swollen. She complained much of heat and profuse perspirations, which returned several times in the twenty-four hours. On the 8th of April the urine was ammoniacal, and contained mucus. The hands were still swollen and erythematous; face flushed; pulse 100, full, as in rheumatism; acid smell of perspiration; respiration 28; movements thoracic and abdominal; abdomen soft; pupils large; nights sleepless. Ordered a grain of opium every six hours, with six ounces of wine daily, and a chop. On April 13th the good effects of the opium and support were very apparent. The patient had passed good nights, and was tranquil in the day. Perspiration lessened. Urine retained in the bladder for thirty-six hours was at length passed voluntarily; it was acid, and without mucus. Tongue pale and moist. The slough on the back had deepened. The pupil still continued large. Occasional contraction of the muscles of the legs. No permanent rigidity.

Hands remained swollen and stiff, but less red. She was unable to move the shoulders freely. On April 22d the hands had recovered their normal appearance, and had lost their stiffness. The legs could be moved more freely. The sense of constriction round the chest was gone; pulse 96; skin cool and dry; appetite good; urine normal, but she could not empty the bladder oftener than once in twenty-four hours. From this date she slowly recovered. The opium was continued throughout her convalescence. At the beginning of June the muscles of the lower extremities were galvanized regularly. By the end of the month she was able to stand without help. Her improvement was uninterrupted, and, in September, she left the hospital quite well.

Remarks.—It is a matter of great clinical interest that lesions of the cord are occasionally attended with an affection of the joints not to be readily distinguished from that which occurs in acute rheumatism. When this happens there may be difficulty in determining the pathology of a case. It may, indeed, be impossible to say whether the symptoms at a certain stage are due to disease of the cord, or to a rheumatic state of the blood. In such instances we have a proof of the near relations of humoralism and solidism; for one observer may maintain that the local lesions have a common origin in the altered state of the blood, whilst another may with equal confidence assert their dependence upon a primary disturbance of the nervous centres. The case here recorded is an example of these difficulties. Fatigue from mechanical labour, acting especially on the lumbar and dorsal portions of the spine in a delicate and anxious subject, appears to have injured the nutrition of the cord. For two years, when much exerting herself, the patient felt pain between the shoulders, and a sense of constriction and coldness round the chest. Paraplegia then suddenly came on, followed by redness, pain, and swelling of the larger joints, as in rheumatism. Together with these symptoms, there were others indicating a rheumatic condition—white, furred tongue; flushed face; hot skin; profuse perspirations, having an acid smell; systolic murmur over left ventricle, &c. Was there here a rheumatic state of the blood induced by the spinal lesion; or was the nervous derangement the result of a rheumatic state? Notwithstanding the labours of morbid anatomists and chemical pathologists, we are not at present in possession of any certain knowledge of what constitutes the rheumatic condition. My colleague Dr. Addison, from his clinical experience, has long drawn attention to the

close connexion between spinal lesions and true rheumatism, but has never developed the idea beyond expressing a suspicion of their relation.

At the time this case was under care the treatment was a subject of much observation. The result was very satisfactory. Whatever might have been the state of the cord, it was clearly induced by fatigue, and was soon followed by sloughing of the integuments. It would not, therefore, admit of depletory measures, but, on the contrary, required a nutritious diet, and wine. Opium was prescribed apparently with great advantage; it allayed nervous irritability, and gave the patient sleep.

The following case is also illustrative of the relation between spinal injury and rheumatic symptoms. The same plan of treatment as above was equally successful. The therapeutical view of this subject is certainly not without the greatest interest. No doubt the texture of the cord has but feeble reparative powers, notwithstanding it has been shown by experimenters on animals, that occasionally, after a transverse section, the parts unite, and the functions are re-established.

CASE XXVIII.—*Concussion of the spine; partial paraplegia; redness and swelling of the wrists and ankles as in acute rheumatism. Recovery.*

W. T—, æt. 38, on the 22d January, 1855, inadvertently stepped backwards into a hole, a few feet deep, and received a concussion of the spine. After a few days he became partially paraplegic, with weak sphincters; and at the same time there came on a diffused redness and swelling of the ankles and wrists. The swelling was not from effusion into the joints, but from œdema of the surrounding tissues. The joints were very painful. The redness and swelling were variable in degree. When most marked they presented the usual appearances of rheumatism, or rather of gout, for the erythema was brighter, and the œdema more distinct than in rheumatism. The hands were equally affected with the ankles, though there was no obvious want of muscular power, nor any affection of sensation in the upper extremities. Tongue clean. Pulse 120. No acid perspirations. Urine high coloured, free from deposits; of normal quantity. The nerves of the surface generally were hyperæsthetic to a slight touch, but deep pressure gave less inconvenience. The treatment consisted of good nourishment, wine and brandy freely administered, and opium to allay pain and overcome sleeplessness. The pulse gradually acquired more power, and sank to 80. The affection of the joints continued in varying degree through March, April, May, and June. From the beginning of April there was an improvement in the power over the legs. The same treatment was continued throughout without the use of mercur-

rials, local depletion, or counter-irritation. In June, the patient was able to walk without assistance. During sleep, the hands and feet, wrists and ankles, often became erythematous and swollen. There was occasional formication in the lower extremities. Sleeplessness, from the beginning of the case, and throughout was a troublesome symptom. In July, the patient was able to leave the hospital, and to resume to some extent his duties as a medical practitioner. He was under the care of my colleague Mr. Cock.

CASE XXIX (Plate IV, figs. A, B, C).—*Anæsthesia of left arm without any other symptom. After three years, gradual loss of muscular power in the arm, with wasting of the muscles; subsequently, a similar affection of the right arm, but in a less degree. Death from general paraplegia at the end of five years from a fall, by which the anterior columns of the cord were ruptured in the lumbar region. Thickening and adhesions of the meninges, especially in the cervical region of the cord; atrophy of the posterior columns, of the posterior roots of the nerves, and of the gray substance, with a development of fibrous tissue.*

Mary S—, æt. 38, a nurse in Guy's Hospital, complained in 1853 of anæsthesia of the left arm, which had come on gradually for nearly a year. There appeared to be entire loss of feeling below the elbow, but, on testing the sensibility upwards to the shoulder and over the scapula, she gave vague and often contradictory answers, at one time affirming, and at another denying, that she perceived impressions made upon the same points of the skin. This discrepancy was perplexing, and led at the time to the belief that her ailment was either feigned or hysterical. The sensibility at the upper part of the thorax, in the axilla, and at the inner part of the arm, was perfect. The muscles were well nourished, the movements powerful and well directed; but the anæsthesia was so complete, that she was unable to hold anything in the hand if her eyes were off it. She often complained of gnawing pains extending down the back, across the shoulders, and into the left shoulder-joint; these pains were increased by the changes of weather. Her symptoms continued unaltered for two years. The following note was made of her case in December, 1855: "Complete anæsthesia limited to the left arm, no wasting of the muscles, no affection of the leg on the same side, general health in all respects good. Electro-contractility of the muscles of the affected arm good. Electro-sensibility greatly diminished. During the next two years there was gradual loss of power, principally in the left shoulder, but also generally throughout the arm, with marked wasting of the muscles. The right arm became at the same time similarly affected, but in a much less degree. She walked quickly, but with a shuffling gait. The left leg was dragged. She was unable to lift the arms over her head, or to extend them horizontally, but when they hung down she could grasp with tolerable firmness and carry heavy weights. She continued to make frequent complaint of pain in the arms and down the back, and of a feeling of weight at the epigastrium. Her manner was often excited, her nights restless, and she was subject to attacks of tremulousness and chilliness like ague, with a sense of

general numbness. About the middle of December, 1857, she accidentally fell forwards upon the stone steps of the hospital, from stepping upon her dress whilst assisting a patient into a cab. Her left temple was cut, and she was rendered insensible by the fall. On recovering consciousness, a short time afterwards, the legs were found to be quite paralysed, and there was almost entire loss of sensation. The weakness of the arms was greatly increased. There was entire loss of sensation below the elbows, and but feeble traces of sensibility above. The muscles were also much wasted. After the accident, the urine became ammoniacal and contained pus. The skin over the sacrum rapidly sloughed, and she died exhausted at the end of a month.

Post-mortem examination.—General wasting of the muscular system. Lateral ventricles of brain dilated and containing clear fluid. The septum lucidum perforated in many places from atrophy. No disease of the bones or ligaments of the spine. The dura mater of the cord was much thickened, apparently by chronic inflammation. This thickening was most marked at the lower part of the cervical enlargement, and along its posterior surface (Plate IV, B, c). In the dorsal region there were plates of true bone, formed by ossific degeneration of the inner layers of the thickened dura mater. One of these plates opposite the third dorsal vertebra was half an inch in length, a third of an inch in width, and a line and a half thick. As these plates were developed by degeneration of the layers of the fibrous membrane, they merely enveloped the cord without producing any pressure upon it. The arachnoid was thickened and opaque, and the two surfaces adherent. In the visceral layer in the lumbar region, several cartilaginous (fibrous) plates. These changes were most marked in the neck, but were continuous down to the cauda equina. The texture of the cord itself had undergone important changes, as shown in Plate IV. About half an inch below the medulla oblongata, on the left side, there was a cyst occupying the position of the gray matter. Its walls consisted of fibrous tissue and compressed nerve-tissue. There was a similar, but smaller cyst, on the right side, at a lower level. No more than a trace of it comes into view in the section drawn (fig. A). The cysts contained colourless limpid fluid. At the cervical enlargement, as seen at A, B, the posterior columns and the gray matter were extremely degenerated. They consisted of some remains of the columns, imbedded in a stroma of fibrous tissue. The posterior roots of the spinal

nerves were included in the degeneration, and the sheaths were thickened in common with the surrounding membranes. The section at *b* shows this. The lower section at *c* did not happen to include the nerve-roots, though the same conditions obtained. The anterior columns and portions of the anterolateral columns were normal, except in the dorsal region, where the anterior columns were ruptured transversely across, apparently at a recent date, and probably by the fall which brought on the fatal symptoms. Viscera of chest healthy. Liver healthy. Acute suppuration of both kidneys; the secreting tissue full of small purulent deposits. Mucous membrane of the pelvis dark coloured and covered with fibrinous exudation. Bladder acutely inflamed; the mucous membrane had sloughed away, scarcely a shred was left on the muscular coat.

Remarks.—The error committed in the early diagnosis of this case was one likely to happen; especially as the patient was a woman. She complained of numbness of the arm. There was nothing visibly wrong with it on the closest examination. The muscles were well developed, the movements were normal, and so were the circulation and temperature. Beside her own account of the numbness there was nothing to indicate disease of the cord or nerves. Her statement, that if she took her eyes off anything held in the hand forthwith she dropped it, was the only circumstance which appeared at the time to have any value as a symptom, and even this was lessened by testing the sensibility. When the patient's head was turned away and she was unable to see what was done, the point of a needle was passed sharply over different parts of the arm. Below the elbow there was an uniform testimony to the absence of all feeling, but upwards there was every kind of contradiction. When she denied feeling at a part, a minus sign was put on it with a pen; when she affirmed it, a plus sign was marked. After mapping out the skin with plus and minus signs, the parts were again tested, and with contradictory results; the plus signs fell over the minus spots, and the minus signs over the spots before marked with plus signs—and so on, in the most uncertain way as often as the trial was repeated. This led to a hasty and false conclusion that the patient was feigning, or that her malady was the vagary of an hys-

terical state. Further clinical observation in other cases, and the examination of the cord in this, have elucidated what was at its early stage so bewildering. When the sensibility of a part is obscure or doubtful, the testimony of the individual as to impressions made upon it may be also doubtful. The same occurs to us with our healthy sensibilities when, conversely, weak impressions are made upon us. When we look at an object scarcely visible, at one moment it appears, and the next is lost. There is in our minds the same discrepancy as to whether we see it or not, as this patient manifested when asked whether she felt or not. Her contradictions were a proof of the obscurity of her sensations, and her convictions fluctuated between certainty and uncertainty, no doubt because the evidence was to her equivocal.

The lesion began apparently in the membranes, and thence extended to the cord, implicating the sensitive roots of the nerves.

There was no history of any acute invasion, nor did the symptoms at any period indicate acute disease.

The dura mater of the brain occasionally offers a similar form of chronic thickening. Though the morbid change must be referred to inflammatory action, the process must have been most gradual; so gradual indeed, that the symptoms were only such as were referrible to atrophy, although the exudation thickened the membranes, and infiltrated the posterior columns. There was no rigidity or other form of spasmodic affection of the muscles, as might have been expected in spinal meningitis.

Whether the exciting cause of the meningeal inflammation was injury, exposure to cold, or a rheumatic condition of the blood, is uncertain. There was no change in the pericardium or valves to corroborate the opinion of its being rheumatic. But, whatever the original cause, its course would be determined by the diathesis of the patient; and hence, in the treatment of such a case, we must determine not only the seat and character of the local lesion, but also view it through the peculiarities of the constitution, whether gouty, rheumatic, scrofulous, or syphilitic. Unless we approach accuracy of diagnosis in both these respects, the therapeutics of the case may be no better directed than the efforts of an engineer, who should

pour medicine down the funnel of his engine, because the power fails in the piston.

It is probable, that at any early period, this case would have been benefited by repeated blisters, and the continued mild use of mercury and iodide of potassium.

The fatal accident was peculiar. The adhesions of the membranes prevented the movements of the cord in the sheath, and exposed it to stretching by any sudden motion of the spine.

The sections of the cord (Plate IV) show to what extent disorganization may take place, and yet the cord serve as a conductor of the voluntary power. The changes at A, B, C, must have been present at the time of the accident, when the patient was able to walk about quickly, and with no more than a shuffling gait, and some dragging of the leg.

CASE XXX.—Pain in back and loins for a year. Profuse hæmaturia, followed after a month by weakness of the legs, which gradually increased to complete paraplegia. Malignant disease of lumbar glands and of the right kidney, extending into the bodies of the vertebræ, and causing sloughing of the cord.

Mrs. W—, æt. 58, a poor needle-woman, overworked, and but scantily fed, was admitted into Guy's Hospital, December 5th, 1857, under the care of Dr. Wilks, for partial paraplegia of the lower extremities. She had been confined to her bed for eight weeks. There was emaciation of the whole body, but especially of the muscles of the legs, which were loose and flabby. She was just able to stand, but not to walk. The back was straight. No abnormal protrusion of any of the spines of the vertebræ. For a year she had had great pain across the loins and back, with some indefinite tenderness. This was at first supposed to be due to her sedentary habits, and then to rheumatism. A month before her legs began to fail her she had profuse hæmaturia, which was thought to arise from calculus in the kidney. After her admission into the hospital the paraplegia gradually became complete, without any preceding rigidity or involuntary jactitation of the legs. The integuments over the sacrum sloughed, and a similar tendency was manifested over the sides of the knees, from one leg resting on the other. She died exhausted January 20th, 1858.

Post-mortem examination.—The outside of the theca vertebralis was covered with a thin layer of grayish offensive pus. The last dorsal and the three upper lumbar vertebræ were infiltrated with cancer extending from the lumbar glands. The body of the first lumbar vertebra was sloughing. The slough-

ing process had thence extended to the adjacent portion of the theca vertebralis, and to the body of the cord, which was ash-coloured, and entirely disintegrated, from the eighth lumbar vertebra, to the filum terminale. Several broad cartilaginous laminae in the lumbar arachnoid. No inflammatory exudation within the theca. Above the eighth dorsal vertebra the cord was remarkably pale and flaccid. No discoverable exudation among the tissue. The right kidney was enlarged by cancerous deposit. Left kidney healthy. Uterus and liver healthy. Cancerous tubera on and under the pleura of both lungs, and cancerous deposit in some of the bronchial glands.

CASE XXXI.—*A wrench of the neck followed after six months by a "stitch" in the neck, supposed to be neuralgic. Extensive development of cancer about the upper dorsal vertebrae; throughout the right lung; up the back of the neck under the deep muscles; and inwards between the laminae of the vertebrae. Paralysis of the arm and right leg. Softening of the cervical portion of the cord. Death sudden.*

Robert P—, æt. 34, a farm-labourer, was admitted under my care, August 5th, 1858, for paralysis of both arms and of the right leg. Intelligence perfect. The account he gave of his illness was, that six months previously he was taken with a "stitch" in the neck under the right ear. The pain "was so bad it almost crazed him." After a short time the pain extended to the left side of the neck towards the occiput, and thence downwards between the shoulders into both arms and into the legs. The pain under the left scapula was for a time very distressing. When he had suffered thus for four months, the left arm began to get numb and powerless from the shoulder downwards. He continued able to walk about very well until three weeks before his admission, when the right arm also and the legs began to fail him. The sphincters retained their power for a fortnight longer. On admission, both arms from the shoulders were powerless, but he could move the fingers slightly. Loss of sensation almost complete throughout both arms. Right leg paralysed, left moved with some freedom. Loss of sensation as high as the fourth intercostal space. Left chest uniformly enlarged and universally dull on percussion, including the sternal region. Heart displaced to the right side. Respiration performed entirely by the right lung. Diaphragm and ribs moving freely on this side. Movements of head and neck without pain. Spine straight. No pain in any part. Respiration 44. Pulse 120. The following day, August 6th, at eleven a.m., the breathing became much embarrassed, and he died quite suddenly at two p.m. After the post-mortem examination the friends gave an account of his having wrenched his neck about a year before in throwing hay into a loft.

Post-mortem examination.—The left chest equally distended,

and the heart displaced to the right side by the development of medullary cancer in the left lung. With the exception of a part of the centre of the lung, the pulmonary issue was entirely destroyed. The pleura was thickened and cancerous, and firmly adherent to the ribs. In the right lung there was a tumour of the size of an orange, having the usual characters of fungus hæmatodes. The cancerous growth had a firm attachment to the anterior part and sides of the body of the third dorsal vertebra, and extended upwards on both sides of the neck, under the deep muscles, as high as the third cervical, and inwards between the laminae, so as to come in contact with the theca vertebralis. The theca was thickened, and the trunk of the fourth cervical nerve invaded on the left side. Unfortunately there was no opportunity to dissect the nerves of the brachial plexus, to determine their relations to the disease outside the vertebral canal. The cervical enlargement of the cord was swollen and softened, and granule-cells were scattered through its tissue. This change had apparently advanced into the cord from the right side of the neck. There was no cancerous deposit inside the theca vertebralis. The arachnoid had its normal appearance. It was the substance of the cord only which had begun to suffer from the proximity of the new growth. Head not examined. Viscera of abdomen healthy.

CASE XXXII.—*Gradual loss of power in right arm, and subsequently in left; after two months and a half, partial paralysis of legs; breathing diaphragmatic; frequent vomiting; pulse quick and feeble. Death by exhaustion, after seven months. Strumous tubercle in the lower half of the cervical enlargement of the cord.*

Elizabeth W—, when eight months old, began gradually to lose the use of the right arm. After a fortnight the left became weak in a similar way. She came under my care as an out-patient at Guy's Hospital, April 13th, 1857, when the paralysis had lasted two months. The wasted arms then hung loose and useless. The head was retracted between the shoulders. The neck stiff. The legs were weak, but could be moved voluntarily. The muscular system generally was wasted, but of the arms most. The skin was constantly warm and freely perspiring. Occasional vomiting. Quick, very feeble pulse. A strumous swelling, the size of a small nut,

was noticed in the skin of the right arm. A distinct history of struma on the father's side. The diagnosis was of tubercular deposit in or about the cervical portion of the cord. At the early part of May the right knee became swollen from effusion into the synovial membrane, and from this date both legs became partially paralysed. There were frequent spasmodic contractions in both legs, but most in the right, which was the weaker. In June the breathing was hurried and entirely diaphragmatic. Vomiting frequent. Difficult deglutition. Diarrhœa. During June and July vomiting and diarrhœa continued. There was great heat of skin. Profuse perspirations. Ammoniacal urine. Pulse 140. Respiration 40. The long muscles of back became atrophied. Shoulders drawn up by the elevator muscles of the scapulæ. There still at this time remained traces of voluntary movements in the legs. She died September 12th from emaciation and exhaustion.

Post-mortem examination.—Only the cervical portion of the cord was allowed for examination. The surrounding structures were healthy. The cord itself, in the lower half of the cervical enlargement, opposite the origin of the sixth and seventh cervical nerves, appeared to be enlarged. This enlargement arose from the presence of a strumous tubercle, which at this part had caused complete absorption of the proper tissue of the cord. This formation seemed to have had its origin in the right posterior and postero-lateral columns, thence extending by successive deposits, until the cord was gradually destroyed, only slight traces of the anterior columns remaining where the tubercle was largest. The chief part of the tumour, from the centre outwards, was opaque, yellow, and friable; it consisted of granules, decaying nuclei, cells, and fat. This opaque dead part was surrounded by a transparent thin layer of more recent exudation, consisting of granules, nuclei, and imperfect fibre-cells, with no free oil-globules. Above the tubercle, the two layers of arachnoid were firmly adherent, and by contraction had constricted the cord. Just below the tubercle, the substance of the cord was so soft that it did not retain its form when unsupported by the membranes.

Remarks.—The gradual onset of the paralysis in this case, and its gradual extension until both arms became paralysed, obviously indicated a progressive organic change in the cord. The nature of this change was also to be plainly inferred from the hereditary tendencies through the father's side, and from the actual presence of a strumous formation in the arm.

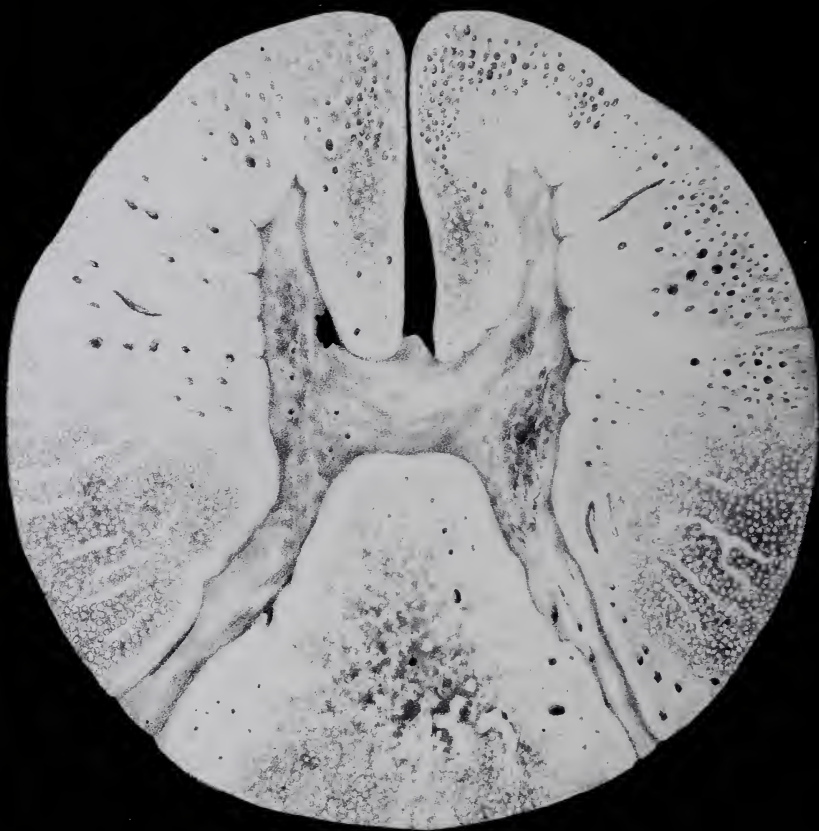
That during the earlier stages of its course the disease should have been one of cervical paraplegia, the power over

the lower extremities continuing after the arms were paralysed, accords with what has been noticed in other cases, but when instead of central disease, the lesion primarily affects the external parts of the cord, at least of the anterior columns, the legs suffer first, and often exclusively if the lesion be moderate.

It is a matter of regret that the state of the sensibility of the legs was not determined. Perhaps, from the age of the child, it could not have been determined.



Plate I.



Bo. 86. 106

Bo. 86. 106. The Queen

PLATE I.

To illustrate Dr. Gull's cases of Paraplegia.

Transverse section of the spinal cord in the dorsal region (Case XVIII), showing atrophy of the gray substance, and inflammatory degeneration of the columns ($\times 12$.)

The atrophy did not affect the caudate vesicles. These, by a higher power, were seen to have their normal structure. The white substance was symmetrically degenerated from chronic inflammation. The exudation cells had undergone fatty degeneration, and were incrustated with fat-globules. The capillaries are seen to be similarly incrustated, producing irregular white lines. The symmetry of the lesion was very exact. It included a small portion of the anterior columns on either side of the anterior fissure, the posterior half of the lateral columns, and the centre and posterior portion of the posterior columns. The part of the posterior columns adjacent to the posterior horns of the gray substance was normal. There was no exudation amongst the gray substance. The apparent traces of such, seen in the drawing, are caudate vesicles.

The artist has not strictly drawn the granule-masses according to scale, but he has faithfully rendered the general appearance of the section under a low power.



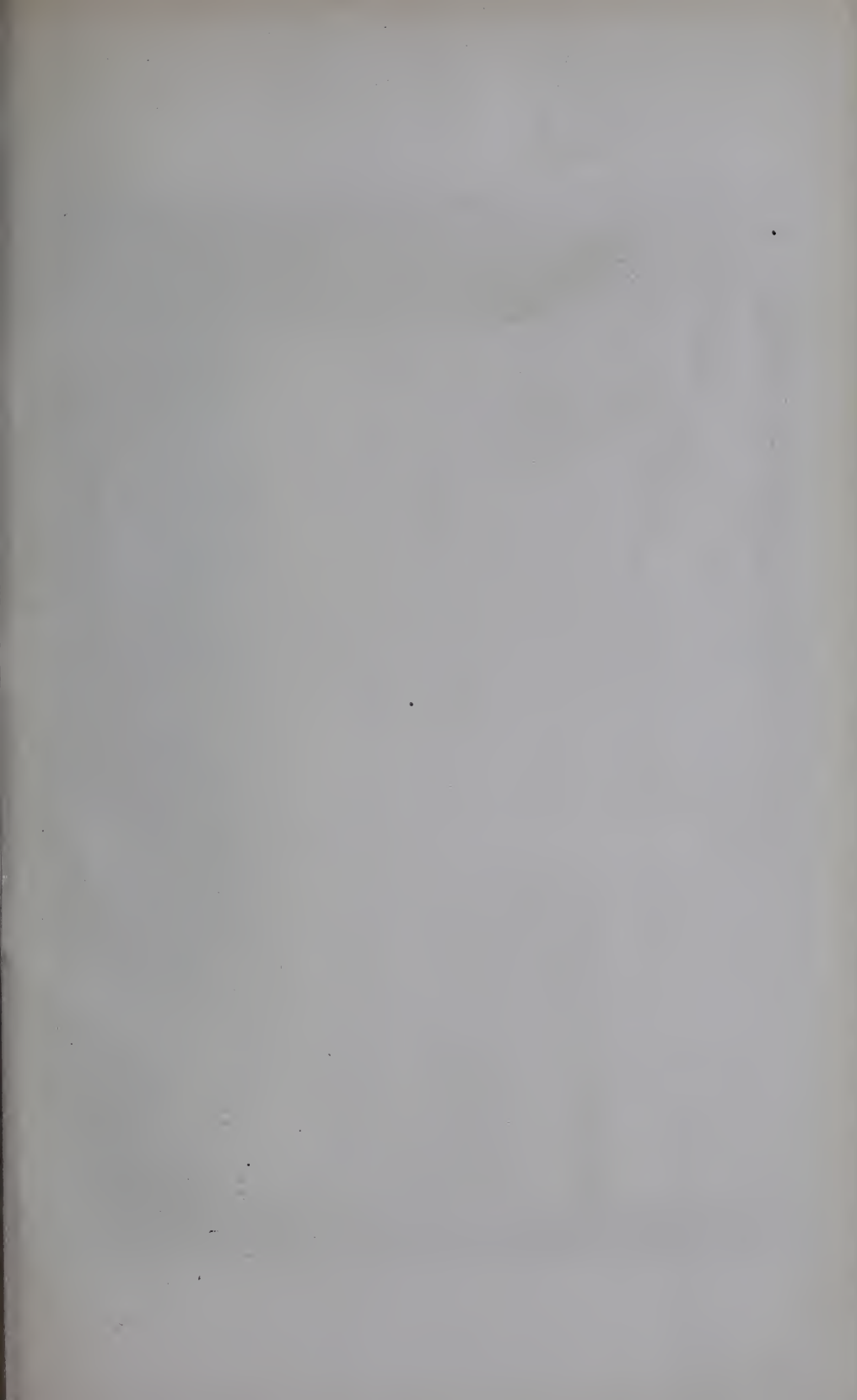


Plate II.



PLATE II.

To illustrate Dr. Gull's cases of Paraplegia.

Longitudinal section of the spinal cord in the dorsal region, from the same subject as Plate I ($\times 12$).

The section was made behind the centre of the cord, so as to pass through the degenerated portions of the lateral columns and through the posterior columns.

As the section is not quite parallel to the axis of the cord, it is only at the lower part of the plate that the section of the posterior columns is shown. At the upper part the knife entered the posterior surface of the gray substance.

In placing the section on the slide, it was slightly torn and the parts displaced, but it was thought best that the artist should faithfully draw the section as put before him.

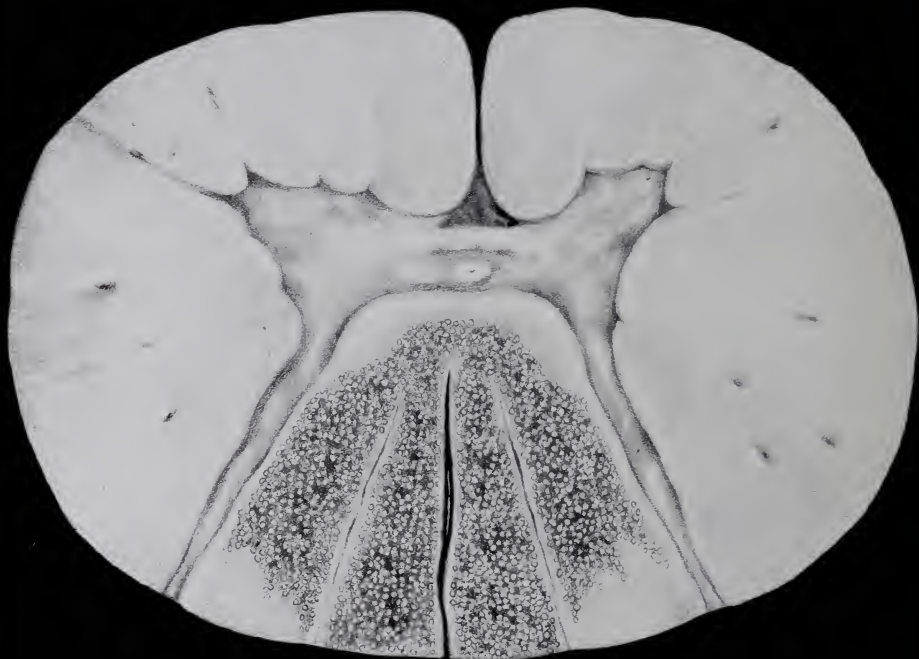
The degeneration of the lateral columns on the right and left of the section, and of the posterior columns in the centre, is but a repetition of that described in Plate I; but here the position of the exudation-cells amongst the tubules of the white substance was more distinct.

At the lower part of the section, on either side of the accidental fissure, it is seen how entirely the gray substance has escaped the exudation, and the same holds in the corresponding portion of gray substance on the right side; in this respect the lateral columns and the small portion of the posterior columns included in the section at the lower part of the plate present a remarkable contrast.

What appearance there is of exudation in the gray substance is due to the presence of normal caudate vesicles.



A



B

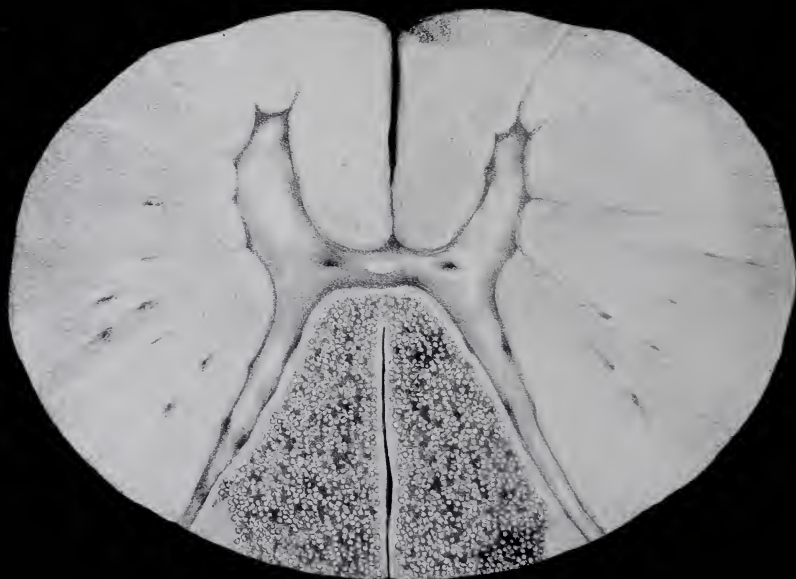
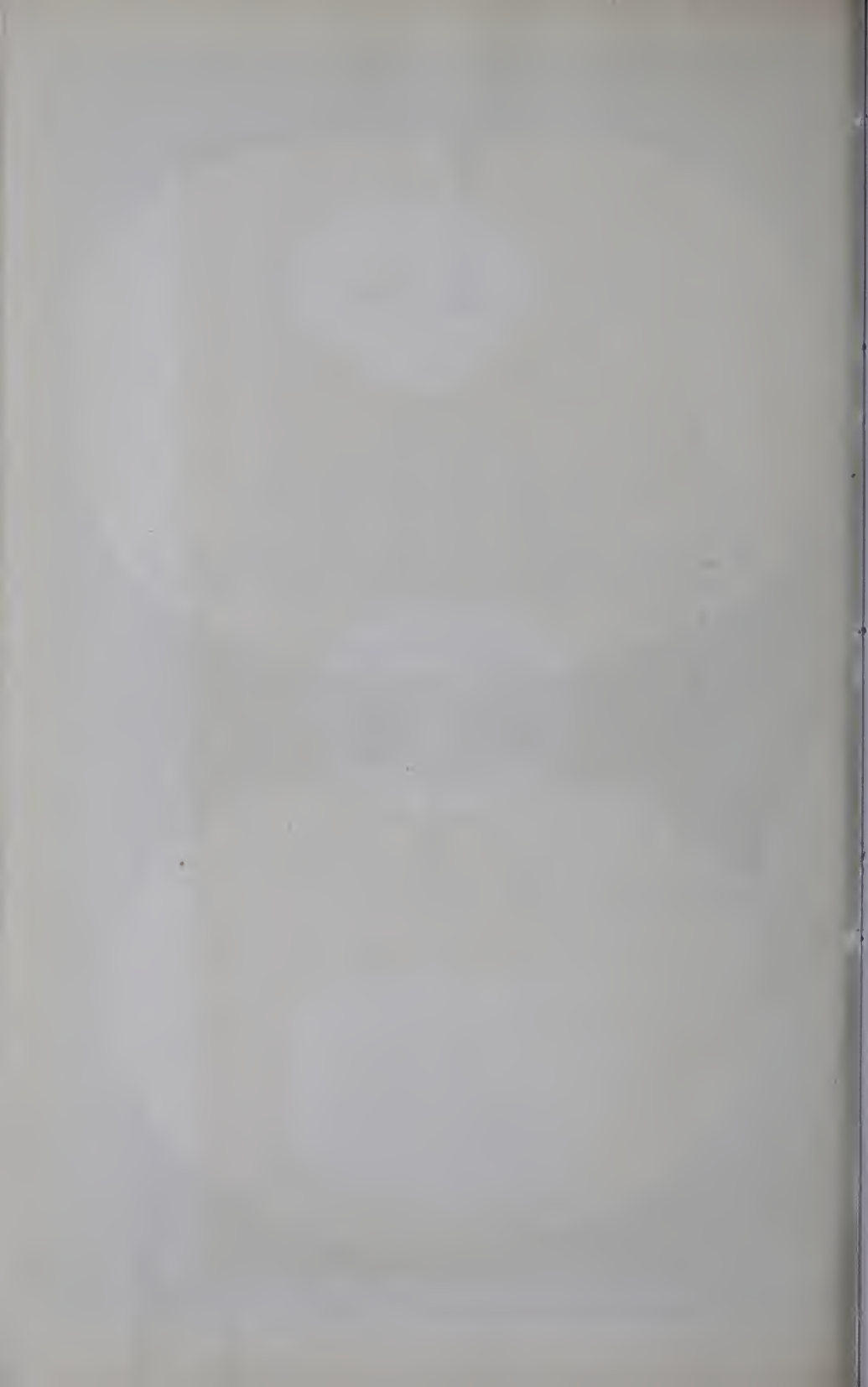


PLATE III.

To illustrate Dr. Gull's cases of Paraplegia.

Transverse section of the cord (Case XIX), showing degeneration of the posterior columns from chronic inflammation ($\times 9$).

The anterior and antero-lateral columns, and the gray substance, were normal. The upper section in the plate (A) is from the upper cervical region; the lower (B) from the lower dorsal region. The granular appearance was due to fatty degeneration of the inflammatory exudation. Though the artist has exaggerated the relative size of the granules to the columns, for distinctness' sake, he has strictly maintained their relative distribution. The lesion was remarkable, from its being so entirely limited to the posterior columns, though it affected them throughout their own length.



A



B



C

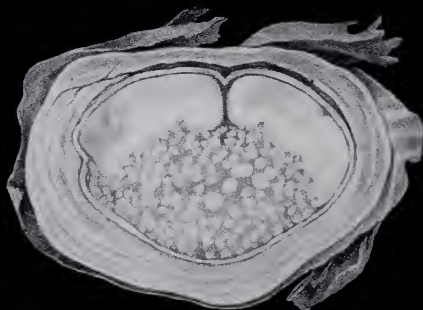


PLATE IV.

To illustrate Dr. Gull's cases of Paraplegia.

Transverse sections of the cord and its membranes, Case xxix
($\times 2\frac{1}{2}$).

A. Section through upper part of the cervical region. The left side of the cord was here distorted by the development of a cyst in the gray substance. A smaller cyst of the same kind existed in the gray substance on the right side, but at a lower level, so that only a trace of it is visible in this section. These cysts had distinct walls of fibrous tissue and condensed nerve-substance. They contained clear colourless fluid.

B. Section through upper part of the cervical enlargement, showing great thickening of the membranes, and degeneration of the posterior columns and gray substance, including also the posterior roots of the nerves, with the development of common white fibrous tissue in place of the normal structures.

C. Section about the middle of the cervical enlargement. The membranes, and especially the dura mater, extremely thickened. This change was greatest on the posterior surface of the cord, where the membranes were adherent together. The posterior columns, the gray substance, and the posterior roots much degenerated. Some of the normal structure of the posterior columns is seen lying imbedded in a stroma of fibrous tissue.

These changes were due to chronic inflammation, apparently advancing from the membranes into the substance of the cord.





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